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ABSTRACT

An extended literature survey and evaluation of neurologic experience (Chile, India, Groote Eylandt/ Australia) was performed to test the assumption that Manganese exposure is the cause of idiopathic Parkinson's Disease or Idiopathic Parkinsonism. There is no support for the contention that these disorders share a common clinical presentation, natural history or outcome. There are key differences which distinguish the demographic features of manganese-exposed and idiopathic parkinsonian populations. The neuropathologic findings in manganic populations routinely spare the substantia nigra and are not associated with Lewy bodies-two hallmarks of Parkinson's Disease. Neither sera levels nor in-situ brain levels of manganese correlate with idiopathic parkinsonian occurrences. The Groote Eylandt syndromes, held to be examples of manganese-associated neurologic disorders (either direct or indirect [as a trigger or synergist]) are separate familial, inherited examples of Olivo-Ponto Cerebellar Atrophy and Type 1 Ehler's Danlos syndrome. There is no support for the assumption that manganism or manganese exposure produces, or is associated with, idiopathic parkinsonism.

It has been noted that "For some time, the motor deficits of manganese poisoning were held to mimic Parkinson's Disease" ([1] Weiss 1990) and that "Parkinsonism may be a form of chronic manganism."([2]Barbeau 1984) The following discussion will identify factors that discriminate between the processes of manganism and idiopathic parkinsonism. The delineation of these factors results from a review of the clinical, demographic, neuropathologic and immunohistochemical features of human and experimental manganism and Parkinson's Disease (idiopathic parkinsonism) identified in over 250 publications and iterated during discussions with neurologists providing care for "manganese intoxicated" patients in Chile (Drs.Mena, Kase), India (Dr. Wadia), Taiwan and Groote Eylandt (Drs. Kilburn, Mathews, Burt). To identify one group of manganese exposed individuals, the author (with Dr. Tim Burt) reviewed the clinical histories of all patients reported from Groote Eylandt and examined a group of afflicted from that location.

Human manganism is a stereotyped disorder. Virtually identical are the clinical descriptions of the affected emerging from Japan, Chile, Italy, Morocco, Mexico, Russia and Rumania. Strikingly similar are reports of neurologic symptoms and signs related to various occupational manganese exposures of miners, ore-processors, welders, battery or electrode manufacturers irrespective of the nature of exposure (Mn particles, aerosols, fumes) through pulmonary, gastrointestinal or skin routes. The clinical disorders are identical for all exposure routes. Unsupported is the contention that small particle size or fume exposure preferentially enters the nervous system through a "nose to brain shunt". There is no support for the contention of manganese orthograde passage along the olfactory nerve, nor transynaptic passage of aerosol manganese or fume manganese (or any metallo toxin). Although manganese, (as with Aluminum, viral proteins, and wheat germ agglutinin) absorbed through the nasal mucosa, and incorporated into the olfactory nerve may be carried to the olfactory bulb, these materials do not enter the basal ganglia through diffusion into the brain substance. Aerosolized Mn likely: A). produces toxicity through pulmonary passage, B). Does not "bypass" this pulmonary system C). Produces neurologic difficulties indistinguishable (in dose, dose:exposure duration, and clinical features) from those associated with other absorption routes. The literature contains no support for the contention that Manganese valence state determines the extent, nature or permanency of Mn-associated neurologic difficulties and it is suggested that "aging of Mn_3O_4 may rapidly produce MnO_2".

The three stages of clinical manganism are without equal either in idiopathic Parkinson's Disease or the fifteen variants of parkinsonism. These stages defined by Mena, Wassermann, Rodier, and Emara include:

1. The early behavioral abnormalities of Locura Manganica which are reversible and follow exposures of 1 to 10 years.
2. After an interval of 2-6 months, there emerge "intermediate" difficulties with varying neurologic symptoms. Varying degrees of reversal of deficits follow therapy with L-DOPA or EDTA chelation.
3. Neurologic deficits affecting sensory organs, coordination, corticospinal tract function and the basal ganglia. There are no descriptions for these deficits merging with, or becoming or resulting in parkinsonism after delays of more than 10 years.

The earliest phase of manganism, "locura manganica" or "manganese madness" is without equivalent in Idiopathic Parkinsonism or its variants. Behavioral disorders, often characterized as "compulsive acts" occur before other manifestations of Manganism. These acts are preceded by anorexia and accompanied by nervousness, irritability ([3 Mena).], impulsivity (singing, running, breaking items)([4] Schuler 1957) with violent components ([3,5] Bronstein 1988)([6] Banta 1977)([7] Wang 1989), hallucinations or terrifying dreams([3,6,7,]). Changes in sexual activity occur (excitability or indifference, impotence, insomnia) and there may occur episodes of euphoria, "uncalled-for laughter" (which is inappropriate in context, extent and its ability to "infect" the afflicted. The laughter persists with a fixed, 'rictus' facial expression), disorders of sleep along with headache in addition to muscle cramps and back pain. Additionally noted are flights of ideas, compulsory acts and verbosity as part of a pattern of emotional instability occurring in over one-half of the afflicted ([4] Schuler 1957). Although depression occurs in one-quarter of Idiopathic Parkinson's disease patients, associated psychiatric or emotional difficulties reflect only the toxicity of medications.

Approximately one-half of the clinical descriptions of the early phase of manganism, allude to the reversibility of the symptoms upon cessation of exposure, chelation with EDTA or therapy with L-DOPA or its equivalent.

Neurologic symptoms during the "intermediate" phase are seldom encountered in parkinsonism. Up to three-quarters of patients develop headache, muscle pains, sialorrhea, altered sweating, and spontaneous abnormalities of sensation (hearing deficits, paresthesia). Up to half of patients have disordered cerebellar function (not seen in Idiopathic Parkinsonism) including

discoordination of arm, hand, leg and gait function in addition to cerebellar nystagmus. In the intermediate stage, features of manganism mirror idiopathic parkinsonian bradykinesia, masked facies, hypophonia and later tremor and gait difficulties. However these superficial similarities vanish as the illness matures or becomes the subject of neurologic scrutiny. The masked facies of manganism, Rodier's "masque manganique" is "jovial and fixed, which gives the patient a dazed appearance". The patient with idiopathic parkinsonism is slack-jawed, without tone, facial expression or spontaneous motion. The bradykinesia of manganism, unlike its mirror illness, includes features of clumsiness and cerebellar disfunction.

With the exception of rare individuals (those with low levels of exposure, and four of the 6 patients from Taiwan, whose facies were immobile, posture was stooped and steps were festernating-features highly suggestive of idiopathic Parkinsonian), the late stage of manganism is characterized by dystonic rigidity involving face (to produce grimace), tongue, neck (torticollis), arm or hand (extensor dystonia) or leg (muscular hypertonia). Gait alterations, unlike the festernation of idiopathic parkinsonism, involve a characteristic "toe stepping" gait (Von Jaksch's "Pas de cog" or "hahnetritt") or "cock walk" (Wadia) to which is added the complications of sensory impairment and cerebellar disfunction. The cock walk performed in marked plantar flexion and leg pronation is often associated with dystonic extension of the great toes. The tremor of manganism, appearing during the intermediate or later stages, is both a resting and action tremor, often of wide amplitude. Movements are described as "flapping" or beating involving the proximal arms and rarely the whole body. The tremor has little of the resting, "pill rolling" quality. The manganese tremor is easily separable (Mena, Kase, Wadia-personal communications) from the resting tremor of idiopathic parkinsonism which is small amplitude, "pill rolling", involving the fingers or wrists. The involvement of the corticospinal tract in manganism is without correlate in idiopathic parkinsons. Faced with the early neuropsychiatric symptoms of Mn toxicity as well as the commonly associated patterns of disordered mood, cerebellar disfunction and hypertonic or dystonic rigidity most investigators (Mena, Barbeau, Klawans, Wadia) have separated clinical Manganism from both idiopathic Parkinsons disease and its variants.

Notably absent from the reports of Manganism are any statements reflecting the evolution of this disorder into self-perpetuating idiopathic parkinsons either in populations with ongoing Mn exposure or those for which exposure has ceased.

RISK FACTORS AND DEMOGRAPHIC EPIDEMIOLOGY

Three lines of evidence separate manganism from idiopathic Parkinsons Disease:

A. The demographic features of individuals with neurologic manganism differ from those of parkinsonism.
B. There are no data reflecting increased incidence of idiopathic parkinsons-increases expected if manganese contributed to the idiopathic parkinsons risk in selected locations or throughout the globe.
C. The neurologic ailments associated with manganese exposure on Groote Eylandt (Australia) are autosomomal dominant disorders for which manganese is without causal relationship.

Prevalence rates for PD include those reported from Sweden (1/1000)([8] Broman 1963), the United States (60-114/100,000)([9] Pratt 1967), Japan (80.6/100,000)([10] Harada 1983), caucasians ((60-187/100,000)([11] Kondo 1986). The incidence rates for the syndrome have not changed in Rochester, Minn. (20/100,000/year in 1945-54 vs 20.5 in 1967-79)([12] Kurland 1958)([13] Rajput 1984) These figures are greater than those reported for Japan (10.2/100,000/year)(10). Comparable figures for manganism are not available. No scientific study of Parkinson's Disease alludes to the contribution of manganese to the predisposition to or rate of occurrence of PD. No PD clusters have been identified in proximity to manganese point sources (mines, smelters, ferrous metal industries, gas additives).

The age occurrence of PD is invariably after fifty years, with cases rare before 30 years (13). The mean age of onset is 64 years (10) in Japan with death occurring five years later. In China the mean age of onset is 51.5 years ([14] Tanner 1989) In the United States the peak incidence appears later than 70 years (10 Rajput 1984)([15] Martilla 1983). These ages of affliction contrast sharply with neurologic manganism fully a decade earlier in appearance. In India, manganism occurred amongst individuals of 20-30 years. Similar ages existed for miners in Chile Morocco and Rumania for whom pre-symptomatic employment was as short as six months and as long as 15 years. Considerable variation in age attack rates, disease severity and presentation reflect individual variation, the nature of exposure (e.g. wet versus dry drilling, mask usage,), exposure duration, and particle size. However the numerous studies of manganism do not define the contribution of other neurologic diseases to manganism predisposition.

Sex differences have not been defined for PD in comparison to the overwhelming preponderance of male sex in neurologic manganism.

There exist no examples of Manganese neurologic toxicity in family members with the exception of husband-wife manganism from skin absorption in Connecticut. As many as 46% of patients with Parkinsonism have afflicted family members ([16] Kondo 1984) and there is an increased rate of the disease in siblings and parents but not

cotwins. ([17] Eldridge 1984)([18] Martilla 1989)

At least 7 case:control studies have evaluated risk factors for the development of Parkinson's Disease. Increased risk has been associated with atherosclerosis, encephalitis ([18] Martilla 1989), rural location, market gardens, chemical and printing plants and wood pulp (14 Tanner 1989). Reduced risk has been associated with ulcer disease, diabetes, cigarette use([16 Kondo 1984]) and low blood pressure. Manganese exposure has not been identified as a risk factor; although other exogenous toxins (MPTP, MPP plus and phenothiazines) have been identified.

The identification of cerebellar disorders among aborigines on Groote Eylandt, Australia forged a link between manganese and neurologic disease. ([19] Kiloh 1980) It had been noted that the aborigines "showed a 'full house' of neurological symptoms...which compares reasonably well with the three stages of manganic poisoning" ([20] Cawte 1991) Afflicted aborigines have been extensively investigated by Drs. C. Kilburn and T. Burt representing the Menzies School of Health Research, Darwin, Australia. These investigations, performed under the auspices of Professor J. Mathews, have not been formally published, and thus will not be iterated in detail. My evaluation of the case data, still photographs and videotaped examinations, has been complemented by the examination of six of the thirty-four afflicted individuals. This group includes 15 members of the BARA moiety centered in the town of UMBAKUMBA and 16 members of the LALARA moiety living in ANGURUGU, Byranybirany, Numulwa, Bickerton, Yirkala and Alayungula (Northern Territories, Aust.) The Bara illness is an example of Type 1 Ehler's Danlos hyperlaxity, inherited through autosomal dominant transmission. The illness, laxity of joints, tendons and connective tissue is associated with scar formation but not with coagulation, lenticular or cardiac abnormalities. No features of cerebellar difficulty have been noted. The BARA disease, commencing in early life, then stabilizes without features of manganism (noted above)) including locura manganica, tremor, dystonia, or pyramidal tract signs. The afflicted have not demonstrated blood manganese levels different from those of other aborigines. Neither manganese nor any exogenous toxin has been identified to serve as a"trigger" or exacerbant of this inherited disorder. Pathologic studies have not been performed.

The LALARA illness is an example of Olivo-Ponto-Cerebellar Degeneration, an autosomal dominant inherited disorder affecting eye movements, speech, and walking. The patients exhibit limitation of upward and lateral gaze (the latter with nystagmus), altered phonation with profound cerebellar ataxia and disordered cortico-spinal tract function. Absent are feature of manganism including personality changes, tremor, "cock-walking" or dystonia. There are no features of Parkinsons disease or parkinsonism amongst these patients. Afflicted patients bear levels of manganese in blood that

are indistinguishable from other Groote aborigines and the degenerative disorder has never been found to be associated with an exogenous toxin such as manganese nor "triggered" by exposure to this material.

Of uncertain origin are three other illnesses seen on Groote-two of which may be transitional forms linking both the Bara and Lalara diseases. Two unconfirmed examples of Amyotrophic Lateral Sclerosis have been reported from the Eylandt and its vicinity. There are no known examples of parkinsonism or Parkinson's Disease ion Groote Eylandt. It has been suggested that Groote Eylandt residents have higher rates of incarceration and criminal behavior than their peers. The Australian commission which investigated this claim identified confounding factors including: alcoholism, petrol sniffing, the high police: population ratio. These are the subject of continuing study.

Summary. Epidemiologic data does not support a relationship between PD or idopathic parkinsons and exposure to manganese. The Groote Eylandt syndromes are examples of autosomal dominant inherited disorders; unrelated to manganese.

THE NEUROPATHOLOGIC STUDY OF PARKINSON'S DISEASE AND MANGANISM

Investigators have summarized the relationship between the pathology of PD and that of Manganism:

> "There is no reason to doubt...that manganese poisoning...produces extensive damage to the brain, involves the globus pallidus, enlarges the ventricles...and spares the substantia nigra, so that Pentschew's argument that it does not resemble paralysis agitans or Parkinson's disease is a good point".([21] Schwab 1968)

> "The clearest basis for distinguishing Parkinsonism and manganism is on histopathological evidence". ([22] Health Assessment EPA)

> These or similar impressions have been shared by Bleecker (1988), Mena and in later presentations by Barbeau himself.

Fewer than ten examples of human manganism have been subjected to neuropathologic examination. These studies support the results of animal experiments by which brain toxicity is induced by manganese of injected, oral and inhalation routes. Degenerative changes are reported([23] Barbeau 1976) in globus pallidus, putamen and caudate nucleus. Less commonly involved are areas of cortex, the pons, the pyramidal tract and anterior horn (in an example of motor system disease) and the red nucleus. The changes include loss of nerve

cells and myelin with reactive glia often present ([24] Yamada 1986). The cerebellar granule cells and the neurons of the thalamus are diminished. The latter study represents one of the few attempts to equate neuropathologic changes with Manganese levels within selected brain areas. Thus nerve cell loss was notable in the globus pallidus, putamen, caudate of a 52 year old male with a 12 year exposure to Manganese while working in an ore crushing plant. The patients brain, examined five years after cessation of exposure revealed no generalized nor localized evidence of increased Manganese compared to brains of non-exposed controls.

Differing neuropathologic changes characterize Manganism in primates (with well pigmented substantia nigra) and rodents (without pigmentation). In rodents, changes affect neuronal cell populations in cortex and cerebellum and only rarely neostriatum. However Chandra in 1972 ([25]) was able to produce changes within the caudate, putamen, substantia nigra and cerebellum of rabbits over one year after tracheal injection of 400mg of MnO2. Using lower doses of aerosolized Mn_3O_4 (11.5-1152 mcg Mn/cu.m per day) Ulrich ([26]) in 1979 was able to detect no pathologic changes in the brains of squirrel monkeys or rats. In this and other studies noted above, rare are the simultaneous determinations of structural cell alterations and loss along with analysis of in-situ Mn levels. Conspicuously unaffected in manganism is the substantia nigra-the site of the most profound changes in most forms of Idiopathic Parkinsons. The cells within the substantia nigra are not depleted in neurologic manganism nor is there evidence of depigmentation of this area, so common in other forms of idiopathic parkinsons. Also absent from all reports of Manganism are Lewy bodies that characterize Parkinsons Disease and many of its variants.([27] Forno 1986). These Lewy bodies are elongated or spherical inclusions associated with nerve cell degeneration within the substantia nigra and the locus caeruleus. Only rarely are these bodies absent within the substantia nigra and locus caeruleus of Parkinson's Disease. The bodies are seen in association with other degenerative diseases of brain but have never been described in association with Manganese intoxication in humans or animals.

The approximately fifteen forms of Idiopathic Parkinsons are characterized by neuropathologic changes within the substantia nigra, locus caeruleus, the nucleus basalis and less commonly the cerebral cortex. These changes include loss of cells, depigmentation in addition to the development of Lewy bodies and changes of cell degeneration including cortical plaques and tangles. Many of these features (involvement of locus caeruleus and substantia nigra, Lewy Bodies, plaques and tangles) represent either rare or undescribed features of either human or experimentally induced Manganism.

SUMMARY. The neuropathologic changes of Manganism differ from those of Parkinson's Disease or its variant by the notable absence, in Manganism, of Parkinson's-associated alterations of the substantia

nigra and the absence of Lewy Bodies.

MANGANESE LEVELS WITHIN BRAIN TISSUE-DO THEY CORRELATE WITH NEUROPATHOLOGIC CHANGES?

If Manganese is associated with the development of PD it would be expected that accumulation of the metal should be noted in sites of major neuropathologic changes in the latter disease.

Few authors ('24 Yamada 1986') have correlated the neuropathologic changes of human manganism or manganese-induced changes in animals to manganese levels within brain tissue. Bonilla in 1982 [28], in a poorly controlled study of "normal brains" found elevated levels of Mn in olfactory bulb (3.36 +/-o.69 mcg/g), pineal gland (4.2 +/- 1.03) in comparison to grey matter (1.54). Similar increases were noted in the pineal gland evaluated by Barbeau in 1976. Larsen [29] (1979) found the highest levels in the putamen (449 ng/g), globus pallidus (395) and caudate (333). Grey matter levels were between 133 and 204 depending on site. Levels were not elevated in the substantia nigra nor pineal and the olfactory bulb was not assessed and there is no mention of neuropathologic studies. Similar studies have been performed in brain tissue (N=3) from patients with Parkinson's Disease [30] (Larsen 1981) revealing Mn levels without increase in putamen (408), globus pallidus (414) and caudate (325). Levels in the substantia nigra were 314ng/g compared to 271 for normal controls. These data are similar to those of Dexter who studied regional Manganese in 7-13 (depending on site) patients with Parkinson's Disease. Levels of 30-50nM Mn/g dry wt. were reported for both afflicted and controls[31] (Dexter 1989) . Elevations of Manganese have not been identified in the brains[32] (Markesbery 1984) or spinal fluid of patients with Alzheimer's Disease [33] (Hershey 1983)-which is seen in relationship to Parkinson's Disease. However increased Manganese levels have been seen in the spinal cord of individuals suffering from motor neuron disease[34] (Mitchell 1986) [35] (Miyata 1983) (perhaps related to the diminished tissue mass of this structure).

SUMMARY. There is no evidence for increased levels of Manganese within sites of major neuropathologic change in Parkinson's Disease.

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Children: Ephraim Paul 10-23-70 (Yale 1992)
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Education:

1963 A.B.	Columbia College
1967 M.D.	Case-Western Reserve Medical School

Postdoctoral Training:

Internship and Residencies:

1967-1968	Intern, Medical Service, Duke University Medical Center
1968-1970	Resident, Neurology, Cleveland Metropolitan General Hospital
1970-1972	Acting Chief, Neurotropic Disease Section, Center for Disease Control, Atlanta, Georgia
1972-1975	NIH Clinical and Research Fellow in Neuropathology, Massachusetts General Hospital
1972-1975	Research Fellow in Immunology, Robert,

Brigham Hospital (Dr. Schur)

Licensure and Certification:

1976	Massachusetts License Registration (36415)
1976	American Board of Psychiatry and Neurology

Academic Appointments:

1975-1978	Instructor in Neurology at Massachusetts General Hospital, Harvard Medical School
1978-1984	Assistant Professor of Neurology, Harvard Medical School
1984-present	Associate Professor of Neurology, Harvard Medical School

Hospital Appointments:

1975-1976	Clinical Assistant in Neurology, Massachusetts General Hospital, Boston, MA
1977-1980	Clinical Associate in Neurology, Massachusetts General Hospital, Boston, MA
1981-1982	Assistant in Neurology, Massachusetts General Hospital, Massachusetts General Hospital, Boston, MA
1986-1988	Associate Neurologist, Massachusetts General Hospital, Boston, MA
1989-	Visiting Physician, DANA FARBER Cancer Center Neurologist, Massachusetts General Hospital, Boston, MA

Other Professional Positions and Major Visiting Appointments:

1975-present	Visiting Neurologist, CHUV (University Lausanne), Department of Neurology (Dr. Regli) and Neuropathology (Dr. Rabinowicz)
1979-present (visiting)	University of Sao Paulo, State University of New York, (Syracuse), University of Maryland, Tokyo University, Duke University, Moffatt Cancer Center (Tampa), New York University Medical Center, Brain Tumor Center (Kansas City), Examiner American Board of Neurologic Surgeons (1982, 1986, 1990)

Editorial Responsibilities:

Editorial Board, CANCER INVESTIGATION
Review responsibilities: New England Journal of Medicine, Jnl. Neuro-Oncol, Neurology, Annals Neurology

Memberships, Offices, and Committee Assignments in Professional Societies:

American Academy of Neurology
American Neurologic Association
The New England Cancer Society
Massachusetts Neurologic Society
International Arts Medicine Association (Board)
Brain Tumor Collaborative Group
Ad Hoc Member Organ System Advisory Committee (Brain), CTEP, NIH,
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Major Research Interests:

1. Neuro-oncology (Primary tumors of the nervous system)
   A. Epidemiology (Glioblastoma, Lymphoma)
   B. Experimental Chemotherapy of Brain Tumors
   C. Viral-Tumor Relationships
   D. Cancer Pain
2. Occupational Neurology
   A. Environmental Neurologic Disorders-Trace Metals, Non-Ionizing radiation
   B. Neurologic Disorders of Musicians
      1). Movement and Dystonic Disorders
      2). Therapy with Botulinum Toxin

Teaching Experience:

Courses: (Postgraduate Harvard)
1. Neurology
2. Neurosurgery
3. Cancer Medicine
4. Neuropathology-Harvard Medical School
5. Neuro-oncology

Visiting Lectureships (1987-present):
International Brain Tumor Symposium (Nikko, Japan; Hakone, Japan; Garda, Italy; Zermatt, Switzerland), Peter B. Brigham Hospital, University of Maine, Lynn Hospital, New England Society of Radiation Therapies, Duke University (Honorary Lectureship), Dana Farber Cancer Center, Emerson Hospital (Honorary Lectureship), St. Lukes Hosp (Milwaukee) (Honorary Lectureship)

Principal Clinical and Hospital Service Responsibilities:
1985- Oncology Research/Education Unit,
Brain Tumor and Cancer Pain Units,
Cox Cancer Center Committee,
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PUBLICATIONS

1. Hochberg F, Nelson N: Human Mercury Exposure. A Symposium. Center for Disease Control, 1971.

2. Hochberg F: Organic Mercury Related Neurologic Disease in Mercury in the Western Environment. Oregon State University, Portland, 1971.

3. Hattwick MAW, Hochberg F, and Landrigan PL: Skunk Associate Human Rabies. JAMA 222: 44, 1972.

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Situ Hybridization in CNS Lymphoma in Immunocompromised Patients. Modern Pathology (submitted January 1990).

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BOOKS

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2. Hochberg FH and Perkin GD: Atlas of Clinical Neurology, Gower Medical, London 1991.

CHAPTERS

1. Hochberg, F.: Prognosis, Contemporary outcomes of Disease, glioblastoma, Chapter 32, Section II, page 128-132, James F. Fries, M.D. and George E. Ehrlich, M.D.(eds.), The Charles Press Publishers, Bowie, M.D. 1981.

2. Hochberg F: Neurologic aspects of systemic tumors. In: Principles of Internal Medicine, Update I, Isselbacher, Adams, Braunwald, Petersdorf, Wilson and Martin (eds.),9th edition, 10th edit, 11 edit. McGraw-Hill Co.,New York, 1981-1990.

3. Takvorian T, Hochberg F, Canellos F, Parker, L., Zervas, N., and Frei, E.: The toxicity of high-dose BCNU with autologous marrow support, in Nitrosoureas: Current status and new developments, Prestakyo, A.W., et al. (eds.), Academic Press Inc., New York, NY, 1981.

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ABSTRACTS (Selected)

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MEMORANDUM

TO: Ethyl Corporation

FROM: Ralph L. Roberson, P.E.

DATE: June 19, 1991

SUBJECT: Impact of Increase in Particulate Emissions on Ambient Air Quality

INTRODUCTION

Based on test data obtained by Southwest Research Institute (SwRI), use of the HiTEC 3000 fuel additive appears to result in a slight increase in average particulate emissions (see Appendix 5). Initial analysis of the SwRI data indicates that the particulate increase varies among car models and driving cycle. The increase ranges from 1.96 mg/mile for the Federal Test Procedure (FTP) to 3.45 mg/mile for the New York Cycle (NYCC). The particulate increase averaged across all vehicles tested and all driving cycles is 2.7 mg/mile. The purpose of this memorandum is to estimate the impact of the increase in particulate emissions on ambient air quality in urban environments. We use two independent approaches, which are described below, to estimate ambient air quality impacts.

ESTIMATE BASED ON SCREAM II RESULTS

In a previous project, Systems Applications International (SAI) used a computer model, the South Coast Risk and Exposure Analysis Model (SCREAM II), to estimate human exposure to manganese that would result in the Los Angeles region from using the HiTEC 3000 fuel additive. Results of the SCREAM analysis are presented in Appendix 13, Attachment 2. SCREAM results indicated that typical 24-hour average manganese concentrations

would be 0.056 $\mu g/m^3$ (see Figure 2, Attachment 2, Appendix 13). This estimate includes a fixed background component of 0.04 $\mu g/m^3$, assumes HiTEC 3000 is used in all unleaded gasoline, and is based on 12 percent of input manganese being emitted. In other words, SCREAM predicts that average ambient manganese concentrations will increase by 0.016 $\mu g/m^3$, if 12 percent of input manganese is emitted. The 12 percent input manganese emission was derived from a measured, average manganese emission rate of 0.25 mg/mile obtained by EPA's Motor Vehicle Emission Laboratory in Ann Arbor. We can estimate the impact of HiTEC 3000 use on ambient particulate concentrations by multiplying the ratio of the increase in particulate emissions (3.45 mg/mile -- SwRI data for NYCC)[1] to the manganese emissions (0.25 mg/mile) times the predicted increase in ambient manganese concentrations (0.016 $\mu g/m^3$). This calculation results in a predicted increase in ambient particulate concentration of <u>0.22 $\mu g/m^3$</u>. Since the SCREAM results are for a typical, 24-hour average impact, we can say that the predicted impact on ambient particulate concentration approximates an annual average. That is, a typical 24-hour average concentration should be about the same as the annual average concentration.

The SCREAM results represent a typical day because SCREAM was run for a computed, average wind speed of 2.5 m/s. SAI examined the meteorological data and determined that the lowest, 24-hour average wind speed for 1981 was 1.21 m/s. If SCREAM were rerun for this minimum average wind speed, the predicted concentration would increase by 2.5 ÷ 1.21 = 2.1. Thus, we estimate that the <u>maximum</u> 24-hour increase in ambient particulate

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[1] Predicted ambient air quality impacts may be adjusted upward or downward as a function of changes in particulate emission rates. For purposes of this analysis, we have used the maximum change in particulate emission rate associated with the use of HiTEC 3000 as reflected in the SwRI test data. Changes in particulate emission rate associated with HiTEC 3000 may actually be lower (see Appendix 5).

concentration would be 2.1 × 0.22 $\mu g/m^3 \approx 0.45$ $\mu g/m^3$. In sum, we observe that our estimated impact on ambient particulate concentrations is based on the highest average increase in particulate emissions, which was determined for the NYCC. If we use the average increase computed for the FTP data (≈ 2 mg/mile), the estimated increase in typical and maximum 24-hour average particulate concentration would be 0.13 and 0.27 $\mu g/m^3$, respectively.

ESTIMATE BASED ON AMBIENT CO DATA

In this approach, we estimate the impact on ambient particulate concentrations by multiplying the ratio particulate emissions to CO emissions times ambient CO concentrations. SAI obtained ambient CO data from EPA's Aerometric Information Retrieval System (AIRS) for four New York City monitoring sites for 1987, 1988, and 1989.

Averaging across years for four New York City monitoring sites, we find the following average annual CO concentrations: 3.08, 3.16, 2.21, and 4.79 parts per million (ppm). The average across the four sites is 3.3 ppm, which is equal to 3.8 mg/m^3. Using the same averaging procedure, we find that the maximum 8-hour CO concentration is 14.4 mg/m^3.

Next, we use MOBILE4 to determine average New York City fleet CO emission rates. When run for an average, wintertime temperature of 31°F and an average vehicle speed of 7.1 mph, MOBILE4 predicts an average CO emission rate of 77 gm/mile. When run for the same speed, but an average annual temperature of 53°F, MOBILE4 predicts an average CO emission rate of 53 gm/mile.

The estimate of the increase in average annual particulate concentration is given by:

$$3.45 \text{ mg particulate/mile} \div 53 \text{ gm CO/mile} \times 3.8 \text{ mg/m}^3 = 0.25 \ \mu\text{g/m}^3$$

The estimate of the increase in maximum 8-hour average particulate concentration is given by:

$$3.45 \text{ mg particulate/mile} \div 77 \text{ gm CO mile} \times 14.4 \text{ mg/m}^3 = 0.65 \ \mu\text{g/m}^3$$

SUMMARY

We have estimated the impact of an increase in particulate emissions as a result of using HiTEC 3000 on ambient air quality using two independent approaches. Based on an increase in average particulate emissions of 3.45 mg/mile (SwRI data for NYCC), our estimate of the increase in average annual particulate concentrations for urban areas ranges from 0.22 to 0.25 $\mu g/m^3$. Yet another estimate of average annual particulate concentrations can be obtained by using the factor presented in EPA's risk assessment document.[2] EPA states that an estimate of ambient air concentration (in $\mu g/m^3$) can be obtained by multiplying vehicle emission rate (in gm/mile) times 30. Applying this factor to a particulate emission rate of 3.45 mg/mile yields: $(3.45 \times 10^{-3}) \times 30 = 0.10 \ \mu g/m^3$. The estimate based on EPA's factor suggests that our estimates based on SCREAM II for the South Coast Air Basin and based on ambient CO data for New York City are reasonable upper bound estimates.

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[2] Comments on the Use of Methylcyclopentadienyl Manganese Tricarbonyl in Unleaded Gasoline, EPA's Office of Research and Development, Washington, D.C., November 1990.

Our estimate of a 24-hour average increase, based on SCREAM II results, is 0.45 $\mu g/m^3$. Our estimate of a maximum 8-hour average increase, based on ambient CO data, is 0.65 $\mu g/m^3$. Since 8-hour maxima must be greater than or equal to 24-hour maxima, we believe it is reasonable to conclude that our two independent approaches yield very consistent results.

RLR/chw

HEAVY DUTY ENGINES AND THE HiTEC 3000 PERFORMANCE ADDITIVE

I. INTRODUCTION

As part of Ethyl Corporation's ("Ethyl") waiver application for use of the HiTEC® 3000 Performance Additive ("the Additive") in unleaded gasoline, Ethyl has reported the results of two separate high speed test programs involving vehicles equipped with V-8 engines, one involving the operation of Ford Crown Victorias for 35,000 miles using fuel containing the Additive (see Docket A-90-16, Docket Entry II-D-3, Appendix 3) and the other involving the operation of Chevrolet Corvettes for 25,000 miles also using fuel containing the Additive (see Appendix 8 to this waiver application). The results of these test programs showed that use of the Additive under severe operating conditions would not adversely affect emission control system operation.

Ethyl believes that these high speed tests were sufficiently severe to be reflective of heavy duty engine operating conditions, and therefore demonstrate that use of the Additive in heavy duty vehicles will not cause or contribute to the failure to meet applicable emission standards. Nevertheless, to investigate further the effect of HiTEC 3000 on heavy duty (in the 10,000 gross vehicle weight range) engines, Ethyl initiated a test program using an engine dynamometer aging schedule similar to those cycles used by manufacturers in engine/catalyst durability testing. A CVS heavy duty dynamometer test cell was not available for this type of test work. Ethyl therefore used normal durability dynamometer test facilities available at ECS/Roush Laboratories ("ECS") in Livonia, Michigan. This dynamometer group was familiar with heavy-duty engine testing and had an emission bench capable of measuring exhaust gas concentrations. In addition, CVS emission testing was completed at Southwest Research Institute ("SWRI") in San Antonio, Texas.

The results of this test program show that use of the Additive will not adversely affect heavy duty engine operation.

II. TEST PROTOCOL

A pair of 1991 5.7 (350 CID) heavy-duty engines were obtained from a General Motors dealer. Accessory engine parts were ordered for these engines and installed at the ECS/Roush dynamometer facility. Current production Ford 5.8 liter (351 CID) engines were purchased for this test, but current emission hardware was not available. Ethyl decided to purchase two 1991 Ford 250 Series pick-up trucks for this program since all emission accessories would be installed in the trucks and be available for dynamometer installation. The Ford engines were removed from the trucks and also installed in the dynamometer.

The engines were operated on a durability cycle similar to those used by engine manufacturers. ECS/Roush selected a cycle

which represents a mix of city and highway truck driving. This 42 step cycle is shown in Appendix Table 1.

Engines were aged in non-electric dynamometers which required that the closed throttle modes, 9 and 17, in the 18-mode emission cycle to be run at 2000 rpm and no load. However, since all engines are operated in the same manner, comparative data is considered valid.

The 18-mode emission test was performed according to the applicable sections of 40 C.F.R. §§ 86.335-79 to 86.346-79. The ECS standard emission cart used a CO analyzer calibrated in the 0-10% CO range for normal non-catalyst engine work.

One engine of each make was aged using Howell EEE while the second engine used Howell EEE plus 0.031 g Mn/gal as HiTEC 3000. All engines were "broken in" using an 18-hour industry type, heavy-duty break-in schedule. A power curve was obtained on each engine and also an 18-mode emission test prior to beginning the durability test work. Emissions were measured after the 18-hour break-in and at approximately 125 and 250 hours. The engines were then removed and shipped to SWRI for CVS emission testing. The change in constant speed emissions from start of test to the 250 hour point provides an indication of both emission differences and deterioration of the catalysts.

In the case of the Chevrolet engines, problems developed, as discussed below, with the placement of oxygen sensors, necessitating additional testing. The Chevrolet test engines accumulated approximately 125 hours of additional operation at ECS after the oxygen sensor problem was corrected.

III. TEST RESULTS

A. Ford V8 Engines

As noted above, emissions were calculated using the 18-mode constant speed data at start of test (after 18-hour engine break-in) and after an additional 250 hours. The results of the 18 mode calculations are shown in Table 1. (Modal data is shown in Appendix Tables 2 through 5).

Table 1

Emission Results
18 Mode Rating Cycle
1991 Ford 5.8 Liter Heavy Duty V8 Engines

Tailpipe Emissions (g/BHP-hr.)

	Start of Test		250 Hours	
	Clear	HiTEC 3000	Clear	HiTEC 3000
HC	0.06	0.08	0.38	0.27
CO	7.1	3.0	18.9	11.3
NOx	6.1	5.8	5.9	5.0

The engines were then removed from the durability dynamometers and sent to SWRI for CVS emission tests. The results of these tests are shown in Table 2.

Table 2

Emission Summary
Ford 5.8 Liter Heavy-Duty Engines
250 Hours Aging Schedule
Tests at SWRI

Tailpipe Emissions (g/BHP-hr.)

Fuel:	Base + HiTEC 3000			Base		
Test No.:	1	2	3	1	2	3
HC	0.24	0.33	0.24	0.29	0.27	0.27
CO	3.65	4.46	4.65	9.61	8.97	8.98
NOx	4.42	4.58	4.60	3.24	3.30	3.41

In general, the 18 mode data shows the same trends as the CVS data. It appears that the MMT engine operates somewhat leaner than the clear engine, CO emission is lower and NOx is higher.

The 18 mode data and the CVS data for both engines at 250 hours is shown in Table 3.

TABLE 3

Emission results
1991 Ford 5.8 Liter Heavy Duty V8 Engines
250-Hour Tailpipe Results (g/BHP-hr.)

	Clear		HiTEC 3000	
	18 Mode	CVS	18 Mode	CVS
HC	0.38	0.28	0.27	0.27
CO	18.9	9.2	11.3	4.4
NOx	5.9	3.3	5.0	4.5

The HiTEC 3000 engine appears to run leaner than the base fuel engine which would result in lower CO emissions and higher NOx levels. However, both engines are giving emission levels which would pass the 110,000-mile standard based on published deterioration factor ("D.F.") factors for this engine family. The D.F. factors for the 5.8L Ford engine are: HC: 1.678, CO: 1.152 and NOx: 1.000. Calculating the emissions at 110,000 miles based on 250 hour CVS data showed that even the high clear engine CO emission will pass the 11.0 g/BHP-hr standard.

These data indicate that there is no detrimental effect of manganese on heavy-duty Ford engine emissions.

B. Chevrolet V8

The Chevrolet engines were run for 250 hours prior to shipment to SWRI for CVS emission testing. Constant speed tailpipe emission tests showed that both engines were similar in their emission characteristics.

The data from the CVS tests at SWRI showed that a problem was occurring in these engines which resulted in higher than expected emission results for both the clear and HiTEC 3000 engines. The CVS data for both engines is shown in Table 4.

TABLE 4

Emission Summary
Chevrolet 5.7 Liter Heavy Duty Engines
250 Hours Aging Schedule
Emission Tests at SWRI (g/BHP-hr.)

Fuel	Base			Base & HiTEC 3000		
Date:	4/29	4/30	5/1	5/4	5/4	5/6
HC	1.42	1.16	1.23	1.30	1.40	1.20
CO	56.23	59.72	62.73	56.54	60.53	55.26
NOx	2.23	2.43	2.36	2.98	3.01	3.06

Analysis of a subsequent 18 mode test showed that emissions were very high on the idle, and low load/speed and closed throttle modes. The engines were returned to ECS/Roush for additional studies. ECS/Roush found that the 02 sensor was cooling rapidly during the low output modes. This cool-off resulted in the computer switching to open loop. The 02 sensor had been located too far downstream of the engine resulting in signal failure at low exhaust flows. ECS/Roush then relocated the sensor to a position which is used in actual vehicle operation. Emission results based on 18 mode data show that the sensor location was critical as seen in Table 5.

TABLE 5

18 Mode Emission Data
1991 Chevrolet 5.7 Liter Heavy Duty V8
Base Fuel Engine (g/BHP-hr.)

	Downstream Location	Engine Y Pipe Location
HC	0.89	0.36
CO	13.1	5.0
NOx	3.2	4.4

The comparison of the summary of the two engines with the 02 sensor located in the Y pipe was shown in Table 6 below.

TABLE 6

Emission Results
18 Mode Rating Cycle
1991 Chevrolet 5.7 Liter Heavy Duty V8 Engines
375-Hour Tailpipe Emissions (g/BHP-hr.)

	Base Fuel	Base Fuel + HiTEC 3000
HC	0.36	0.21
CO	5.0	3.9
NOx	4.4	3.8

The 18 mode data for these two engines at 375 hours is shown in Appendix Tables 6 and 7. The data in Table 6 show results similar to the Ford engines and indicate that the use of HiTEC 3000 does not adversely affect emission results.

The Ford engines were also operated for 250 hours prior to shipment to SWRI for CVS emission testing. The Ford data for the 18 mode emission tests showed no problems with the Ford 02 sensor

location, even though these sensors also had been located downstream of the Y pipe for durability test work. This is because the Ford engines use an electrically heated sensor, which is not dependent upon sensor location.

Emission data at 375 hours were then obtained for both engines using the 18 mode procedure. These results are shown in Table 6.

IV. CONCLUSION

There is no indication based on these tests that the General Motors or Ford heavy duty engines are adversely affected by the use of HiTEC 3000.

APPENDIX TABLE 1

Heavy Duty Durability Schedule
ECS/Roush Dynamometer
Two minutes per Step

Step	RPM	Torque	Step	RPM	Torque
1	Idle	No Load	22	3280	54
2	1000	268	23	3600	220
3	1000	60	24	3600	207
4	Idle	54	25	Idle	54
5	2200	268	26	Idle	No Load
6	2200	142	27	1880	54
7	Idle	54	28	1880	67
8	2600	169	29	2200	207
9	2600	135	30	2200	150
10	Idle	No Load	31	3040	268
11	3350	148	32	3040	207
12	3350	135	33	3280	220
13	Idle	54	34	3280	54
14	Idle	No Load	35	3600	220
15	1880	54	36	3600	207
16	1880	67	37	Idle	54
17	2200	207	38	Idle	No Load
18	2200	150	39	1000	268
19	3040	268	40	1000	60
20	3040	207	41	Idle	54
21	3280	220	42	2600	300

APPENDIX TABLE 2

18 Mode Emission Data
Tailpipe After Break–in

Engine: Ford 5.8L
Serial No. A13078, Base Fuel

Mode	HC, PPM	% CO	NOx, PPM
1	66	0.04	28
2	19	0.04	917
3	8	0.00	1022
4	12	0.04	913
5	8	0.04	266
6	13	0.06	1022
7	73	0.48	421
8	0	0.00	805
9	5	0.02	117
10	4	0.07	936
11	5	0.01	922
12	6	0.04	931
13	4	0.03	255
14	7	0.04	957
15	51	0.39	367
16	0	0.06	832
17	2	0.06	127
18	3	0.03	106

HC: 0.06 g/BHP—hr.
CO: 7.1 g/BHP—hr.
NOx: 6.1 g/BHP—hr.

APPENDIX TABLE 3

18 Mode Emission Data
Tailpipe after Break–in

Engine: Ford 5.8L
Serial No. A13079, Base Fuel + HiTEC 3000

Mode	HC, PPM	% CO	NOx, PPM
1	151	0.04	16
2	36	0.05	1051
3	6	0.05	1365
4	17	0.06	796
5	16	0.07	272
6	20	0.07	853
7	69	0.12	638
8	12	0.07	795
9	7	0.05	97
10	6	0.07	878
11	10	0.06	1313
12	3	0.06	813
13	5	0.04	314
14	12	0.04	915
15	67	0.11	633
16	6	0.06	728
17	0	0.04	105
18	132	0.01	31

HC: 0.08 g/BHP–hr.
CO: 3.0 g/BHP–hr.
NOx: 5.8 g/BHP–hr.

APPENDIX TABLE 4

Simultaneous 18 Mode Emission Data
Ford 5.8L Engine at 250 Hours
Base Fuel

Mode	HC, PPM	CO %	NOx PPM
1	464	0.64	42
2	132	0.01	859
3	91	0.02	1476
4	91	0.00	832
5	66	0.00	252
6	91	0.14	602
7	141	1.32	352
8	98	0.03	653
9	62	0.01	169
10	104	-0.04	831
11	75	0.00	1481
12	88	0.02	725
13	64	0.02	224
14	97	0.01	780
15	145	1.50	333
16	84	0.01	691
17	63	0.03	159
18	430	0.03	40

HC: 0.38 g/BHP—hr.
CO: 18.9 g/BHP—hr.
NOx: 5.9 g/BHP—hr.

APPENDIX TABLE 5

Simultaneous 18 Mode Emission Data
Ford 5.8L Engine at 250 Hours
Base Fuel + HiTEC 3000

Mode	HC PPM	CO %	NOx_2 PPM
1	437	0.72	56
2	70	0.05	1059
3	78	0.07	1269
4	70	0.05	832
5	81	0.06	785
6	76	0.02	857
7	132	0.75	362
8	88	0.03	723
9	56	0.10	90
10	94	0.07	780
11	78	0.02	633
12	88	0.05	773
13	54	0.04	155
14	81	0.05	965
15	129	0.61	408
16	79	0.02	991
17	45	0.06	70
18	204	0.07	32

HC: 0.27 g/BHP—hr.
CO: 11.31 g/BHP—hr.
NOx: 5.04 g/BHP—hr.

APPENDIX TABLE 6

Simultaneous 18 Mode Emission Data
Chevrolet 5.7L Engines at 375 Hours
Base Fuel

Mode	HC PPM	CO %	NOx_2 PPM
1	135	.10	23
2	96	.09	23
3	70	.12	201
4	115	.10	32
5	239	.10	24
6	133	.07	22
7	60	.05	2232
8	102	.09	34
9	261	.11	24
10	118	.07	43
11	62	.12	201
12	104	.13	33
13	276	.08	23
14	116	.07	26
15	64	.06	2217
16	147	.10	28
17	301	.09	26
18	324	.11	9

HC: 0.36 g/BHP—hr.
CO: 4.99 g/BHP—hr.
NOx: 4.44 g/BHP—hr.

APPENDIX TABLE 7

Simultaneous 18 Mode Emission Data
Chevrolet 5.7L Engine at 375 Hours
Base Fuel + HiTEC 3000

Mode	HC PPM	CO %	NOx_2 PPM
1.	500	.04	9
2	115	.03	81
3	81	.06	149
4	79	.02	67
5	142	.02	27
6	84	.01	90
7	55	.02	1850
8	84	.02	81
9	93	.01	46
10	91	.02	87
11	75	.05	85
12	85	.01	79
13	129	.02	25
14	92	.02	87
15	75	.02	2000
16	88	.01	71
17	78	.03	0
18	130	.03	83

HC: 0.21 g/BHP—hr.
CO: 3.94 g/BHP—hr.
NOx: 3.8 g/BHP—hr.

REFORMULATED GASOLINE AND THE BENEFICIAL EFFECTS OF HiTEC 3000

In materials submitted by Ethyl Corporation ("Ethyl") to the U.S. Environmental Protection Agency ("EPA" or "Agency") in connection with Ethyl's May 9, 1990 waiver application, Ethyl showed that use of the HiTEC 3000 Performance Additive ("the Additive") could have a beneficial effect on the reactivity of vehicular hydrocarbon emissions and, therefore, ambient ozone concentrations.[1/] Since submission of this data, Congress has enacted amendments to the Clean Air Act which establish a new reformulated gasoline program. Under the new program, gasoline must be reformulated to achieve at least a fifteen percent reduction in the emission of volatile organic compounds and certain toxic air pollutants.[2/]

The attached tables draw from the speciation emission data previously presented to EPA to illustrate that use of the Additive should make it easier for refineries to comply with the new reformulated fuel requirements. The tables show that use of the Additive in a wide spectrum of fuels reduces non-methane hydrocarbons (2-14%), relevant toxic emissions (i.e., benzene, formaldehyde, 1,3 butadiene, and acetaldehyde, which are reduced in the aggregate 13-31%), and reactivity (23-30%) when compared to fuels of equal octane.

1/ See In Re Application for a Fuel Additive Waiver Filed by Ethyl Corporation under § 211(f)(4) of the Clean Air Act (May 9, 1990), Appendices 4 and 5; Comments in Support of the Waiver Application for the HiTEC 3000 Performance Additive (July 23, 1990), Appendix 1.

2/ CAA § 211(k)(3).

Table 1

Comparison of Speciated Emissions
From Two 1988 Ford Crown Victorias

Fuel	Octane Enhancer	FTP-HC(1) gm/mi	Reactivity(2) Ozone, gm/mi	CAA Regulated Toxics(3) gm/mi
Howell EEE	HiTEC 3000 (Car F-3)	0.512	0.605	0.0182
	Xylenes (Car F-5)	0.568	0.863	0.0248
	Difference (Xylene - HiTEC)	0.056	0.258	0.0066
	% Improvement with HiTEC	10%	30%	27%
Texaco	HiTEC (Car F-3)	0.494	0.651	0.0148
	Xylenes (Car F-5)	0.554	0.857	0.0215
	Difference (Xylene - HiTEC)	0.060	0.206	0.0067
	% Improvement with HiTEC	11%	24%	31%
EC-1	HiTEC 3000 (Car F-3)	0.567	0.644	0.0165
	Xylenes (Car F-5)	0.563	0.836	0.0189
	Difference (Xylene - HiTEC)	(0.004)	0.192	0.0024
	% Improvement with HiTEC	(1%)	23%	13%

Notes: (1) FTP-HC: Federal Test Procedure Hydrocarbons

(2) Product of the FTP-HC number and "reactivity" factors developed by Dr. William P.L. Carter (University of California, Riverside). Dr. Carter's factors have been used by the California Air Resources Board.

(3) Data shown are aggregate emissions of four toxics (benzene; formaldehyde; 1,3 butadiene; acetaldehyde). The 1990 Clean Air Act requires that the aggregate emissions of five specified toxics (the aforementioned four plus POMs) from 1995 reformulated gasoline be reduced 15% vis a vis the toxic emissions from 1990 "baseline" fuel.

TABLE 2

Comparison of Speciated Emissions and Regulated Toxic Emissions from two 1988 Ford Crown Victorias

Fuel	FTP-HC gm/mi	Reactivity Ozone, gm/mi	CAA Regulated Toxics (1) gm/mi
Howell EEE with HiTEC 3000 added (Car F-3)	0.512	0.606	0.01855
Howell EEE "neat" (Car F-5)	0.595	0.845	0.0256
Difference ("neat" minus HiTEC fuel)	0.082	0.239	0.00705
% Improvement with HiTEC	14%	28%	28%

Note: (1) Data shown are aggregate emissions of four toxics (benzene; formaldehyde; 1, 3 butadiene; acetaldehyde). The 1990 Clean Air Act requires that the aggregate emissions of five specified toxics (the aforementioned four plus POMs) from 1995 reformulated gasoline be reduced 15% vis a vis the toxic emissions from 1990 "baseline" fuel.

Summary of Speciation Analysis on Two Ethyl Fleet Cars
gm/mile

Diff = HiTEC 3000 blend minus Xylene blend (2)

		Howell EEE (2) With Added			Commercial Gasoline (2) With Added			Reformulated Gasoline (2) With Added		
		Xylenes	HiTEC	%Diff.	Xylenes	HiTEC	%Diff.	Xylenes	HiTEC	%Diff.
	HC	0.568	0.513	(10%)	0.554	0.494	(11%)	0.563	0.567	0.7%
	Non Methane HC	0.448	0.387	(14%)	0.442	0.362	(18%)	0.415	0.407	(2%)
	Aromatics	0.090	0.051	(43%)	0.066	0.040	(39%)	0.051	0.028	(45%)
	NO_x	1.373	0.940	(32%)	1.200	0.862	(28%)	1.406	0.982	(30%)
	CO	2.208	1.380	(38%)	2.377	1.496	(37%)	2.472	1.607	(35%)
	Alkenes(C2-C6)	0.047	0.039	(17%)	0.051	0.045	(12%)	0.073	0.063	(14%)
(1)	Benzene	0.016	0.011	(31%)	0.011	0.007	(36%)	0.009	0.008	(11%)
(1)	Formaldehyde	0.006	0.005	(17%)	0.007	0.005	(29%)	0.007	0.006	(14%)
(1)	1,3 Butadiene	0.0007	0.0005	(29%)	0.0011	0.0010	(9%)	0.0012	0.0011	(8%)
(1)	Acetaldehyde	0.0021	0.0017	(19%)	0.0024	0.0018	(25%)	0.0017	0.0014	(18%)
(1)	Toxic Aggregate	0.0248	0.0182	(27%)	0.0215	0.0148	(31%)	0.0189	0.0165	(13%)

NOTES:

(1) These toxics, plus POMs, must be reduced, in aggregate, by 15% in 1995 reformulated gasoline vis a vis levels in 1990 "baseline" fuel. (Per 1990 amendments to the Clean Air Act)

(2) For each of the three type fuels (Howell EEE, Commercial, Reformulated), the octanes of the respective HiTEC blends (0.03gm Mn/gal) equalled those of the respective Xylene blends.

A-91-46
II-D-04

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July 22, 1991

Mr. William K. Reilly
The U.S. Environmental Protection Agency
401 M. Street S.W.
Washington, D.C. 20460

Dear Mr. Reilly:

We understand that Ethyl Corporation has recently resubmitted a new waiver application to the United States Environmental Protection Agency to allow the use of Ethyl HiTec 3000 performance additive in unleaded gasoline. Clark Oil supported the approval of the original Ethyl Corporation submission for this waiver and we continue to support approval of this waiver.

Our studies show, as we have said before, that Clark Oil and other refiners would be able to increase gasoline production by 1-4 percent by either reducing reformer severity or by blending more low octane components such as natural gasoline. In either case the aromatic concentration of gasoline would be reduced by 2-4 percent. Since the average refinery produces approximately fifty percent gasoline from a barrel of crude oil, crude oil requirements for the same gasoline production would be reduced by 2-8 percent. Reducing reformer severity in refineries also saves energy and would result in fewer refinery emissions of sulfur dioxide, nitrous oxides and carbon dioxide.

The 1990 clean air act has, among other things, mandated reductions in Benzene and aromatics in gasoline. In order to achieve these reductions while maintaining suitable gasoline octane levels for todays automobiles, refiners will have to spend billions of dollars to reformulate gasoline. Therefore, the use of this Ethyl additive in gasoline could save the industry and hence the general public millions, or perhaps billions, of dollars on unneeded, new refinery construction.

We urge that the EPA give very serious consideration to the approval of this waiver application and allow all refiners to take advantage of this octane promoting additive.

We would like to thank you for the opportunity to express our views on this very important topic.

Very truly yours,

Jerry Garbutt
President